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CREOSOTE - HUMAN EXPOSURE

EXECUTIVE SUMMARY

Creosote is applied by occupational handlers only. Since it is a restricted-use pesticide that can only be applied by certified applicators or someone under their direct supervision, it is not available for sale to or use by homeowners. A recent voluntary cancellation of all non pressure treatment uses restricts creosote to commercial and industrial settings.

The amount of creosote handled in a given day among pressure treatment facilities depends on such factors as the size of the facility and the number of treatment cylinders on site. In a given facility, the amount of creosote handled per day varies depending on the wood conditioning techniques used for a given charge, on the type of wood being treated, and the type of product being produced (e.g., marine piling vs utility poles).

This chapter is a revision of the earlier draft Human Exposure RED Chapter for Creosote completed on January 27, 2000. EPA received an extensive creosote-specific handler/post-application exposure study from the Creosote Council II that was completed on January 30, 2001. The study was reviewed and accepted by EPA. The new study as well as the earlier pilot study (completed July 2, 1998), estimated worker exposure to pressure treated wood. The new study entitled "Assessment of Potential Creosote Inhalation and Dermal Exposure Associated with Pressure-Treatment of Wood with Creosote" was submitted by the Creosote Council II for all tasks involving pressure treating wood with creosote. As a result of these new data, many of the scenarios and exposures in the earlier draft chapter were revised to incorporate the new data.

The earlier draft chapter contained exposure data from the pilot study which indicated that the workers at the pressure treatment plant were the treatment operator, treatment supervisor, pressure treatment loader, test borer, environmental compliance operator, loader man, and locomotive driver. The exposure of the treatment operator and treatment supervisor were used to represent handler exposure and the pressure treatment loader, test borer, environmental compliance operator, loader man, and locomotive driver were used to represent post-application exposure in the earlier Human Exposure RED Chapter. However, worker titles in the new exposure study were much different although the job functions were similar. Workers included the treatment operator, treatment assistant, cylinder area loader, cylinder area helper, checker, drip pad loader, load-out area loader, load-out area loader helper, forklift operator, oil unloader, test borer, and water treatment system operator. For

this chapter, the treatment operator and treatment assistant are used to represent handler exposure during the pressure treatment process. Exposure data for the cylinder area loader, cylinder area loader helper, checker, drip pad loader, load-out area loader, load-out area loader helper, load-out area forklift operator, oil unloader, test borer, and water treatment system operator are representative of occupational post-application exposure.

The scenarios using the Pesticide Handlers Exposure Database(PHED) and the Chemical Manufactures Association (CMA) data from the earlier draft chapter (completed January, 27, 2000) have been deleted based on the voluntary cancellation of all non pressure treatments. Uncertainties and limitations exist in available exposure data and toxicity information. Specific uncertainty concerns are summarized in Section 10 of this document.

The results of the handler exposure and risk assessment indicate that the creosote inhalation exposures exceed the level of concern for all handler scenarios. The target margin of exposure (MOE) is100 or more and the MOEs are 10 for the treatment operator and 17 for the treatment assistant even with the use of current engineering controls. For dermal risks, only long-term exposures exceed the level of concern for the treatment operators. In addition, cancer risks for all handler scenarios exceed the level of concern (1E-04) for occupational handlers. Most estimated cancer risks are in the range of 1.0E-03 or greater.

The results of the post-application occupational exposure and risk assessment indicate that the creosote inhalation exposures exceed the level of concern for all post-application scenarios. For dermal risks, some of the short-, intermediate- and long-term exposures also exceed the level of concern. The target MOE is 100 or more for short- and intermediate-term risks and 300 for long-term dermal durations. In addition, cancer risks for all post-application occupational scenarios exceed the level of concern (1E-04); all are greater than 1E-03, except for load-out area loader helpers who had cancer risks ranging from 4E-04 to 8E-04.

In a recent submission dated November 25, 2003, the registrants submitted a probablistic worker risk assessment for creosote. This probablistic assessment has been included in the public docket. The methodology and data inputs in this recent submission will be reviewed during the public comment phase.

No chemical-specific data for residential post-application exposure were submitted. Therefore, exposure could not be estimated. Data were not adequate for use in the exposure assessment.

The following table shows the scenarios and the source of data used to develop these scenarios for this preliminary risk assessment.

Table 1. Summary of the Occupational/Nonoccupational Exposure Scenarios and Source of Data

Exposure Scenario	Source of Data
Occupational Handler	
(1a) Mixing/Loading/Applying Liquids at a Pressure Treatment Facility (treatment operator)	Exposure Study Data from Creosote Council II and PHED, 1997used as a surrogate
(1b) Mixing/Loading/Applying at a Pressure Treatment Facility (treatment assistant)	Exposure Study Data from Creosote Council II
Occupational Postapplication	
(1) cylinder area loader operator	Exposure Study Data from Creosote Council II
(2) cylinder area loader helper	Exposure Study Data from Creosote Council II
(3) checker	Exposure Study Data from Creosote Council II
(4) drip pad labor	Exposure Study Data from Creosote Council II
(5) load-out area loader operator	Exposure Study Data from Creosote Council II
(6) load-out area loader helper	Exposure Study Data from Creosote Council II
(7) load out area forklift operator	Exposure Study Data from Creosote Council II
(8) oil unloader	Exposure Study Data from Creosote Council II
(9) pressure treatment test borer	Exposure Study Data from Creosote Council II
(10) water treatment systems operator	Exposure Study Data from Creosote Council II
(11) railroad worker	No data
(12) pole installer	No data
Non-Occupational (e.g., Residential)	
(1) homeowner incidental ingestion and dermal contact with soil contaminated with creosote (e.g., soil contaminated by creosote treated telephone poles) (child)	No data
(2) outdoor homeowner dermal contact with industry pressure treated wood products (e.g., utility poles, posts, shingles, fencing, lumber, piers, etc.) (adult)	No data
(3) outdoor homeowner hand-to-mouth and dermal contact with industry pressure treated wood products (e.g., utility poles, posts, shingles, fencing, lumber, piers, etc.) (child)	No data

4. OCCUPATIONAL AND RESIDENTIAL EXPOSURE AND RISK ASSESSMENT

Occupational and Residential Toxicological Endpoints

An occupational and/or residential exposure risk assessment is required for an active ingredient if (1) certain toxicological criteria are triggered and (2) there is potential exposure to handlers (mixers, loaders, applicators, etc.) during use or to persons entering treated sites after application is complete. For creosote, both criteria are met.

On April 1, 1999, the Hazard Identification Assessment Review Committee evaluated the toxicology data base of Creosote and selected the toxicological endpoints for short-term, intermediate-term, and long-term occupational/residential exposure risk assessments and for carcinogenicity screens. An acute and chronic Reference Dose (RfD) was not selected, as there are no food uses for creosote (USEPA, 1999). An acute or chronic dietary risk assessment is not required for creosote, as there is no anticipated dietary exposure to creosote (USEPA, 1999).

4.1 Occupational / Residential Exposure — Dermal

Dermal Absorption: A 50% dermal absorption value is used for short and long-term, non-cancer assessments. The value of 50% dermal absorption was obtained by comparison of the oral and dermal LOAELs from the developmental toxicity study in rats (MRID # 43584201) and the 90-day dermal toxicity study in rats (MRID # 43616101) using the P1/P13 blend. The oral LOAEL of 175 mg/kg/day observed in the developmental toxicity study, when compared to the dermal LOAEL of 400 mg/kg/day observed in the dermal toxicity study, yields an absorption factor of 44%, which was rounded up to 50% by the Committee, taking into account the dermal irritation which also occurs from dermal exposure to creosote (USEPA, 1999).

Short-Term Dermal (1 day - 1 month): An oral maternal NOAEL of 50 mg/kg/day, based on decreased body weight gain during the study, was chosen for this endpoint. Although a 90-day dermal toxicity study was available, the developmental toxicity study was chosen for the following reasons: 1) dermal toxicity studies (including the 2-week range-finding studies) did not measure developmental endpoints, which are present in both developmental toxicity studies; and 2) the results of dermal toxicity studies would not be protective of infants and children from residential exposure to creosote. An uncertainty factor (MOE) of 100 is applied to this risk assessment (USEPA, 1999).

In a developmental toxicity study using P1/P13 creosote (MRID # 43584201), pregnant female Sprague-Dawley rats (30/dose) were administered P1/P13 creosote at dose levels of 0, 25, 50, and 175 mg/kg/day on gestation days 6 through 15 inclusive. Decreased body weight and food consumption were observed at the 175 mg/kg/day dose level in this study in maternal rats. Decreased uterine weight was observed in maternal rats at the high dose, which is reflected partly by the decreased live fetuses per litter at the high dose (although mean fetal weight was not affected). Cesarean section observations showed significantly increased resorptions and post-implantation loss as well as decreased number of live fetuses per litter at the 175 mg/kg/day dose. Based on the results of this study, the Maternal NOAEL is 50 mg/kg/day, and the Maternal LOAEL is 175 mg/kg/day, based on decreased body weight gain during the study (USEPA, 1999).

No treatment-related malformations (external, visceral or skeletal) were observed in any of the fetuses at 25 mg/kg bw/day. At 50 mg/kg bw/day, the overall incidence of malformations on a fetal and litter basis were statistically elevated compared to controls. However, these individual malformations were not seen at higher dose levels and/or fell within the range of historical control data. At 175 mg/kg bw/day there was (i) an overall significant increased incidence of developmental malformations, (ii) increased incidence of cardiovascular, vertebral and digital malformations, compared to lower dose levels, concurrent controls or historical controls (2429 and 2898 fetuses examined viscerally and skeletally respectively) and (iii) an increased incidence of malformations at this dose level in spite of increased fetal loss (resorptions) (Beck and Lloyd, 1963) thus resulting in fewer fetuses available for teratogenic examination. Although the incidence of fetal malformations observed at 175 mg/kg bw/day dose level in rats was low and could be related to maternal stress (decreased body weight gain and food consumption), the teratogenic potential of P1/P13 Creosote cannot be ruled out. Based on these data, the developmental toxicity LOAEL can be determined as 175 mg/kg/day, with the developmental toxicity NOAEL as 50 mg/kg/day (USEPA, 1999).

Intermediate-Term Dermal (1 month to 6 months): A dermal NOAEL of 40 mg/kg/day, based on decreased body weight gain in males at 400 mg/kg/day, is selected for this endpoint. An uncertainty factor (MOE) of 100 is applied to this risk assessment (USEPA, 1999).

In a 90-day dermal toxicity study (MRID # 43616201), Charles River rats (10/sex/dose) were given dermal applications of P2 creosote in corn oil at dosage levels of 0, 4, 40 or 400 mg/kg bw/day. There was no mortality observed in this study at any dose level. Body weight in high dose males was decreased 7-8% during weeks 9-12 of the study, and body weight gain decreased 15% in high dose males for the treatment period. Food consumption in high dose males was decreased during weeks 2-4 and week 6 by 4-10% vs control. Only slight dermal irritation was observed in high dose males. No effects

were observed on hematology or clinical chemistry. Treated skin in the 400 mg/kg/day dose groups (male and female) was observed with increased incidence of dermal inflamation. Based on the results of this study, the systemic LOAEL is 400 mg/kg/day, based on decreased body weight gain in male rats. The systemic NOAEL is 40 mg/kg/day. For females, the NOAEL is set at 400 mg/kg bw/day since no systemic toxic effects were noted in any of the treated groups (USEPA, 1999).

Long-Term Dermal (greater than 6 months): A parental oral LOAEL of 25 mg/kg/day, based on decreased pre-mating body weight, was selected for this endpoint. An extra uncertainty factor of 3x is applied for use of a LOAEL in this study for occupational risk assessments (USEPA, 1999).

In this study, Charles River Crl:CD rats, 26/sex/group, were dosed by gavage with P1/P13 creosote in corn oil at doses of 0, 25, 75, and 150 mg/kg/day. Pre-mating treatment phase lasted approximately 17 weeks, which may have contributed to the decreased fertility observed in this study. Systemic effects observed in this study for parental animals included decreased body weight during the pre-mating period at all dose levels, with a dose-response noted for this effect. Salivation was also observed at 75 mg/kg/day and above in the F1 generation. Effects in offspring included a dose-related decrease in growth of offspring of the F0 generation starting at 25 mg/kg/day (as shown by decreased pup weight). For the F0 pups, mean number of liver pups per litter was decreased at 75 and 150 mg/kg/day, and percent live pups at 175 mg/kg/day was also decreased. In the F1 pups, the percent live pups was decreased at 75 and 150 mg/kg/day, but pup growth was affected only at 150 mg/kg/day as shown by decreased mean pup weight. Decreased fertility and pregnancy indices were observed in the F1 female parental rats at all dose levels, but this was not interpreted as a treatment-related effect, as it was more likely related to the fact that the critical weight for fertility was exceeded by the 17-week pre-mating interval. Based on the results of this study, the Parental Systemic NOAEL is < 25 mg/kg/day, and the Parental Systemic LOAEL is 25 mg/kg/day, based on decreased pre-mating body weight. The developmental NOAEL in this study is < 25 mg/kg/day, and the developmental LOAEL is 25 mg/kg/day, based on a dose-related decrease in pup body weight for the F0 pups from days 14-21. The reproductive NOAEL is < 25 mg/kg/day, and the reproductive LOAEL is 25 mg/kg/day, based on reduced pregnancy and fertility indices in F1 female parental rats (USEPA, 1999).

4.1.2 Occupational/Residential Exposure — Inhalation (any time-period):

An NOAEL of 0.0047 mg/L, based on decreased body weight gain, altered hematology and clinical chemistry, and increased absolute and relative weight of the liver and thyroid observed at 0.048 mg/L, was selected for this endpoint (USEPA, 1999).

In a 13-week inhalation toxicity study (MRID # 43600901), 20 Sprague-Dawley rats/sex/group were treated for 5 days/week, 6 hours/day with P2 Creosote CTM via whole body exposure at doses of 0, 4.7, 48 or 102 mg/m^3 (0, 0.005, 0.048 or 0.102 mg/L) in air measured gravimetrically. The aerosol size MMAD was between 2.4 and 2.9 microns with a geometric standard deviation between 1.85 and 1.91. Subsequent to the exposure period 10 rats/sex/group were allowed to recover from treatment for 6 weeks (USEPA, 1999).

During the exposure period, two animals (low dose female; mid dose male) were sacrificed in extremis and the cause of morbidity was not related to treatment. Significant treatment-related findings in mid and high dose animals included decreased terminal body weight and body weight gain (m/f), altered hematological parameters (decreased hemoglobin content, hematocrit, erythrocyte and platelet counts; increased reticulocyte counts and mild poikilocytosis, m/f) and biochemical parameters (increased serum cholesterol levels, m/f). In both sexes macroscopic discolouration of the lungs persisted through the recovery period and correlated with the presence of black pigment granules within alveolar macrophages. Both sexes showed increased absolute and relative liver and thyroid weights and increased lung/trachea/body weight ratios. Absolute and relative thyroid weights of high dose animals actually increased after the recovery period. An increased incidence of lesions of the nasal cavity epithelium (chronic inflammation) was noted following treatment (all treatment groups, m/f) but appeared to lessen in incidence and severity during the recovery period (mainly the high dose group, m/f). During exposure an increased incidence of thyroid follicular epithelial cell hypertrophy occurred in all male groups including control and in the high dose female group. At recovery the male incidence remained similar to that observed at exposure while the incidence in females of the high dose group had declined. The incidence of thyroid follicular cell hypertrophy was slightly increased in low and mid dose females after the recovery period. Slightly increased incidence of mild poikilocytosis was observed in all treatment groups (m/f) including the low dose group and control, which persisted through the recovery period. Low dose animals exhibited lesions of the nasal cavity epithelium which had resolved after the recovery period. Based on the results of this study, the systemic LOAEL is 48 mg/m^3, based on decreased body weight and weight gain, altered haematology ad clinical chemistry, increased absolute and relative weight of the liver ad thyroid, and increased incidence of lesions of the nasal cavity. The systemic NOAEL is set at 4.7 mg/m^3 (0.0047 mg/L) for P2 Creosote CTM in rats (USEPA, 1999).

The short-intermediate, and long-term NOAEL of 1.2 mg/kg/day was calculated by converting the inhalation NOAEL of 0.0047 mg/L in Sprague-Dawley rats. The inhalation endpoint of 0.0047 mg/L was converted to an oral equivalent dose using Equation 1 of the HED Route to Route Extrapolations memo dated October 9, 1998 (USEPA, 1998a) presented below:

Equation 1:

$$\text{Inhalation NOAEL (mg/kg/day)} = \frac{\text{NOAEL (mg/L) x RV} \left(\frac{\text{L}}{\text{hr}}\right) \text{x D x A x AF}}{\text{BW}}$$

where:

NOAEL	=	No-observed-adverse-effect level (NOAEL) (0.0047 mg/L)
RV	=	Respiratory volume (10.26 L/hr)
D	=	Duration of daily animal exposure (based on a 6-hour/day study)
BW	= (0.236	Mean body weight in kg of Sprague-Dawley rat kg)
A	=	Absorption - the ratio of deposition and absorption in the respiratory tract compared to absorption by the oral route, assumed to be 1
AF	=	Activity factor - animal default is 1

Carcinogenicity Screen: Cancer information was not initially presented in the initial draft of the hazard identification report (USEPA, 1999). EPA suggested using a cancer slope factor of 7.3 $(mg/kg/day)^{-1}$ for this assessment (Personal Communication, 1999).

The carcinogenicity data base for creosote as required by the Agency in the 1988 DCI consist of a six-month dermal oncogenicity study of creosote conducted in mice. Creosote in this study was tested both as an initiator (5 dermal applications per week for 2 weeks at doses of 500 μg/mouse, 25 mg/mouse, or 56 mg/mouse followed by TPA for 26 weeks) and as a promotor (DMBA as a positive initiator at 50 μg/mouse followed by twice weekly applications of creosote at the same doses as used for the initiation

protocol). As an initiator, creosote did not produce any increase in incidence of benign tumors, but at the 25 and 50 mg doses, squamous cell carcinomas were observed in 2/30 mice at each dose. As a promotor in DMBA-initiated mice, creosote produced dose-related increases in skin papillomas, keratoacanthoma, squamous cell carcinoma, and basal cell carcinoma at the 25 and 50 mg doses. Increases in these tumor types were also observed when creosote was used as both initiator and promoter. This study shows that creosote acts most effectively as a promoter but also functions as a "complete" carcinogen (USEPA, 1999).

Mutagenicity: In consideration of the available evidence that creosote is a positive mutagen, the Agency waived the requirement for the standard mutagenicity battery, and instead required dominant lethal testing of both the P1/P13 and P2 blends. The executive summaries of these studies are shown below (USEPA, 1999).

In a rat dominant lethal assay with P1/P13 creosote (MRID not available), male Sprague-Dawley rats were treated orally once per day for five consecutive days with Creosote P1/P13 at target doses of 725, 362.5 or 181.25 mg/kg body weight/day in a volume of 2.5 mL/kg. Actual doses determined by chemical analysis were 857.5, 330.5 and 230.8 mg/kg/day. Twenty-one rats were dosed at the two lower doses and 26 rats at the highest dose. The vehicle was corn oil. Seven days after the initial dosing, each male was mated with two untreated females per week for 10 weeks (USEPA, 1999).

Creosote P1/P13 was tested to toxic doses. A preliminary toxicity test was conducted with Creosote P1/P13 concentrations of 625, 1250, 1875 and 2500 mg/kg/day for five consecutive days. All rats in the top three dosage groups were dead within three days. No treatment related deaths occurred in the lowest dosage group although other clinical signs were seen following dosing including decreased activity, increased salivation, diarrhea and anogenital staining. In the dominant lethal study, all rats in the top two treatment groups but none in the low dose group showed decreased activity following dosing and all rats in the high dose group had dyspnea. Two animals in the low dose group and two in the high dose group had material around the nose and mouth. Other pharmacotoxic signs were limited to a few animals in the high dose group and included lacrimation, deposition of the test material around the eyes, increased salivation and anogenital staining. One high dose rat died following the fourth dose. A dose-related decrease in body weight in the low-, mid-, and high-dose animals, compared to the solvent controls, was seen during the dosing period, and this initial weight loss was not recovered in mid- and high-dose rats during the ten week mating period. Females were sacrificed 13 days after the midweek of the presumptive mating day and the following data collected: total implantations per female, corpora lutea per female, pre-implantation losses per female, live implantations per female, dead implantations per female, proportion of females with one or more dead implantations, proportion of females with two or more dead

implantations and dead implantations/total implantations (expressed as a percentage). The fertility index, computed as the number of fertile females (with corpora lutea present) per number of mated females, was also determined. Statistically significant differences from control values were seen in a number of endpoints throughout the study; however, with the exception of results from mating group nine, none were endpoints indicative of a dominant lethal effect. In mating group nine, statistically significant increases were seen in dead implantations per female, the percentage of females with ≥ one implantation, the percentage of females with ≥ two implantations and the percent dead implantations per total implantations. These increases were seen at the low and mid doses but not at the high dose. Also, the vehicle control values in mating group nine were unusually low compared to those in the other weekly mating groups (there were fewer preimplantation losses (0.85 per female) and fewer dead implantations (0.41 per female) than seen for the vehicle controls in the other mating groups (1.53 ± 0.37 and 0.85 ± 0.20 per female, respectively) and values for percentage of females with ≥ one and two dead implantations and the percent dead implantations per total implantations were depressed). The results, although statistically significant, are thus not considered biologically significant. Positive and solvent control values were appropriate except where noted for the vehicle controls in mating group nine. There was no evidence that Creosote P1/P13 induced dominantlethals in any germ cell stage in male rats as tested in this study (USEPA, 1999).

In rat dominant lethal assay with P2 creosote (MRID not available), male Sprague-Dawley rats were treated orally once per day for five consecutive days with Creosote P2 at target doses of 775, 387.5 or 193.75 mg/kg body weight/day in a volume of 2.5 mL/kg. Actual doses by chemical analysis were 866.3, 431, or 199.3 mg/kg body weight/day. Twenty-one rats were dosed at the two lower doses and 26 rats at the highest dose. The vehicle was corn oil. Seven days after the initial dosing, each male was mated with two untreated females per week for 10 weeks (USEPA, 1999).

Creosote P2 was tested at an adequate dose. All rats in all treatment groups showed decreased activity following dosing. Other clinical signs, limited to a few animals in which there was a back-up of test material during dosing, were lacrimation, deposition of the test material around the eyes and increased salivation in one high-dose male, labored breathing in one low-dose male and two high-dose males, and material around nose and mouth in two low-dose, one medium-dose and four high-dose males. Reduced food consumption was seen in all high-dose rats. No dosing- or test material-related deaths occurred during the study. A dose-related decrease in body weight in the low-, mid-, and high-dose animals, compared to the solvent controls, was seen during the dosing period, and this initial weight loss was not recovered in mid- and high-dose rats during the ten week mating period. Females were sacrificed 13 days after the midweek of the presumptive mating day and the following data collected: total implantations per female, corpora lutea per female, preimplantation losses per female, live implantations per female,

dead implantations per female, proportion of females with one or more dead implantations, proportion of females with two or more dead implantations and dead implantations/total implantations (expressed as a percentage). The fertility index, computed as the number of fertile females (with corpora lutea present) per number of mated females, was also determined. Statistically significant differences from solvent control values ($p \leq 0.05$) were seen for a number of endpoints during the first nine weekly mating intervals but, with one exception, not in endpoints considered indicative of dominant lethality. The one exception was a significant increase in the number of dead implants over the solvent control value in the sixth mating group at the lowest Creosote P2 dose. This increase was not considered biologically relevant because no significant increases were seen at higher doses or in other endpoints concerning dead implants. In the tenth mating group (exposure to the test material at the spermatogonial stem cell stage), an apparently dose-related increase was seen in the number of dead implantations per female, the percentage of females with ≥ 1 dead implant, the percentage of females with ≥ 2 dead implants and the percentage of dead implantations per total implantations. The increases reached statistical significance at the highest dose for the first two endpoints. Lack of a dominant lethal effect in the eighth and ninth mating groups, which also test spermatozoa that were exposed at the spermatogonial stem cell stage, may possibly indicate treatment-related cell cycle delay. Positive and solvent control values were appropriate.

There was no evidence of a dominant lethal effect in the first nine weeks following treatment; however, significant differences between the control group and the treated group were seen in week ten with respect to dead implantations per female, the percentage of females with ≥ 1 dead implantation and the percentage of dead implantations per total implantations (USEPA, 1999).

4.1.3 FQPA Considerations

As there are no existing tolerances or other clearances for residues of creosote in food, an FQPA assessment is not necessary. Potential post-application exposures to residents, including children (e.g., from use of railroad ties by homeowners), could not be assessed due to lack of exposure data. The available evidence on developmental and reproductive effects of creosote was assessed by the Health Effects Division (HED) Hazard Identification Assessment Review Committee on April 1, 1999 The committee expressed concern for potential infants and children's susceptibility of creosote, based on the severity of offspring vs. maternal effects observed with testing of creosote in the P1/P13 blend developmental toxicity study in rats at the 175 mg/kg/day dose level as well as deficiencies observed in the 2-generation reproduction toxicity study in rats.

Although there are no current Agency guideline neurotoxicity studies available for creosote, the existing studies on creosote indicate no evidence of neurotoxicity for either the P1/P13 or P2 blends of creosote (ATSDR, 2002). Based on the above, and realizing that creosote is currently registered only for non -food use and is a restricted use pesticide, no additional neurotoxicity testing will be required at this time.

4.1.4 Acute Toxicology Categories

Table 1 provides the acute toxicity categories for creosote. It also provides the results of the toxicity tests.

Table 1. Acute Toxicity Categories for Creosote

Test	Results	Toxicity Category
Acute Oral Toxicity	LD_{50} = 2,451 mg/kg (M); 1,893 mg/kg (F)	III
Acute Dermal Toxicity	LD_{50} > 2,000 mg/kg	III
Acute Inhalation Toxicity	LC_{50} > 5 mg/L	IV
Primary Eye Irritation	Irritation clearing in 8-12 days	II
Primary Dermal Irritation	Erythema to day 14	III
Dermal Sensitization	Study unacceptable	NA

NA - Not applicable, no toxicological endpoint.

4.1.5 Summary of Endpoints of Concern

Endpoints for assessing occupational and residential risks are presented in Table 2. The results of the exposure tests are also provided.

Table 2. Endpoints for Assessing Occupational and Residential Risks for Creosote

Test	Results	MOE
Acute Dietary Exposure	Not required	NA
Chronic Dietary Exposure - Reference Dose (RfD)	Not required	NA
Short-term Dermal Exposure (1 to 30 days)	Oral developmental rat study NOAEL 50 mg/kg/day based on maternal effects + 50% dermal absorption	100
Intermediate-term Dermal Exposure (1 to 6 months)	Dermal 90-day dermal toxicity study in rats NOAEL 40 mg/kg/day based on decrease in body weight gain	100
Long-term Dermal Exposure (greater than 6 months)	Oral two generation reproduction study in rats LOAEL 25 mg/kg/day based on decreased pre-mating body weight + 50% Dermal absorption	300
Short-, Intermediate-, and Long-term Inhalation Exposure	NOAEL 0.0047 mg/L based on decreased body weight gain, altered hematology, and increased weight of liver and thyroid (converted to 1.2 mkd)	100

Test	Results	MOE
Oral Cancer Slope Factor*	7.3 $(mg/kg/day)^{-1}$+ 50% dermal abs.	NA

NA- not applicable, no toxicological endpoint.

* Slope factor is for benzo(a)pyrene, a component of creosote, and used as an indicator of carcinogenic potential of creosote.

The short-term, long-term, and cancer endpoints are based on toxicity endpoints from oral studies. A dermal absorption rate of 50 percent was applied to oral exposure estimates to establish risks reflective of a dermal endpoint as recommended in the report of the Hazard Identification Assessment Review Committee (USEPA, 1999). The intermediate-term dermal endpoint is based on the results of a dermal study. Thus, no dermal absorption rate is required.

4.2 Occupational Handler Exposures and Risks

EPA has determined that there are potential exposures to mixers, loaders, applicators, and other handlers during typical use-patterns associated with creosote and from use in commercial and industrial settings. The following types of handler exposures have been identified:

(1a) mixing/loading/applying liquids at a pressure treatment facility (treatment operator);
(1b) mixing/loading/applying liquids at a pressure treatment facility (treatment assistant);

Table 3 provides a description of exposure scenarios for occupational handlers.

Table 3. Exposure Scenarios for Occupational Handlers

Exposure Scenario	Scenario Description
(1a) Mixing/loading/applying liquids at a pressure treatment facility (treatment operator)	Scenario pertains to a wood pressure treatment plant. Liquid ready-to-use creosote is prepared from concentrate and loaded into the retort using a mechanical pump. Exposure occurs while pumping liquid into the retort and pumping liquid from retort back into holding tank. Exposure data from the Creosote Council II study for the treatment operator (TO) were used in this assessment (Creosote Council II, 2001). TOs operated and monitored application system valves and controls, they sometimes opened and closed cylinder doors, and they supervised the insertion and removal of charges (loaded dried, debarked poles or untreated ties) of poles from the treatment cylinders.
(1b) Mixing/loading/applying liquid formulation at a pressure treatment facility (treatment assistant)	Scenario pertains to a wood pressure treatment plant. Liquid ready-to-use creosote is prepared from concentrate and loaded into the retort using a mechanical pump. Exposure occurs while pumping liquid into the retort. Exposure data from the Creosote Council II study for the treatment assistant (TA) were used in this assessment (Creosote Council II, 2001). TAs performed many of the same functions as the TOs and sometimes assisted the TO in charge preparation, cylinder cleaning and maintenance, filter cleaning, mixing of treatment solution, and also participated in some loader operations moving charges.

Creosote is used by occupational handlers only. Since it is a restricted-use pesticide that can only be applied by certified applicators or someone under their direct supervision, it is not available for sale to or use by homeowners. Creosote is now to be used exclusively in industrial settings.

4.2.1 Handler Data and Assumptions

In the course of development of this risk assessment, data from the chemical-specific handler study was exclusively used to assess potential risks to workers at pressure treatment facilities.

The Agency used a worker exposure study on pressure treatment use submitted by the Creosote Council II to provide chemical-specific handler dermal and inhalation exposure data in support of the re-registration of pressure treatments of creosote (Creosote Council II, 2001). These data were used to support scenarios 1a and 1b of the assessment. These data were reviewed internally by EPA (USEPA, 2001).

No dermal exposure studies were identified in the available literature. This is consistent with EPA statements in the most recent re-registration position document entitled *Wood Preservative Pesticides: Creosote, Pentachlorophenol, and Inorganic Arsenicals: Position Document 4* (USEPA, 1984). "There are no quantitative data on dermal exposure to these workers." Since EPA currently wishes to pursue estimation of quantitative exposures to creosote, the Creosote Council II's 2001 study was used in order to estimate dermal exposure information for occupational handlers exposed during the pressure treatment process.

Inhalation exposure data were also sparse. In the document entitled *Wood Preservative Pesticides: Creosote, Pentachlorophenol, and Inorganic Arsenicals: Position Document 4* (USEPA, 1984), EPA states that "the Agency still has no definitive data on the identity of the airborne component chemicals of creosote to which workers are exposed in wood treatment plants where creosote is used." In addition, there are apparently no exact methods to develop inhalation exposure data through personal monitoring of creosote.

Because of the overall variability in the composition of creosote (e.g., over 100 known chemicals are components of creosote), it is difficult to characterize its exact nature. Since neither the characterization of airborne creosote or the development of inhalation sampling

methods are specific for creosote, there exists a high variability in the creosote inhalation data presented in the literature. Most of the studies presented in the literature were conducted by industrial hygienists using methods approved by the National Institute for Occupational Safety and Health (NIOSH) and Occupational Safety and Health Administration (OSHA) for polycyclic aromatic hydrocarbons (PAHs), phenols/creosols, and the individual constituents of the PAHs (i.e., naphthalene, phenanthrene, anthracene, etc). Since the Creosote Council study is the most recent study presented on creosote exposure and presents both dermal and inhalation exposure, it was used to provide exposure estimates. Other studies found in the available literature are also presented in the post-application section.

The Creosote Council II study was used to provide actual dermal and inhalation exposure information for occupational handlers exposed during the pressure treatment process. This chemical-specific information is believed to provide a more accurate estimation of actual exposures than surrogate data currently available from PHED or CMA. Since the actual mixing/loading/applying of creosote is an entirely mechanical process, dermal and inhalation exposures to the handlers that participate in the pressure treatment process (e.g., treatment operators and **treatment assistants**) were used to estimate total handler exposure during the pressure treatment process. These handlers are selected because they are representative of the population of handlers that would be loading the wood preservative and operating the retort. A description of their duties is provided in Table 3. The study examined exposure to handlers for four to five days. **The geometric mean concentrations reported for the treatment operator in those four to five days were used as an estimate of handler exposure. The geometric mean concentration was used to represent a typical exposure over time**.

Creosote Council II 2001 Study Synopsis: **The 2001 Creosote Council II study was conducted to determine the dermal and inhalation exposure of workers exposed to creosote while performing routine tasks related to pressure treatment of lumber, utility poles, and railroad ties at four typical commercial treatment facilities in the U.S. and Canada, per the requirements of the U.S. Environmental Protection Agency, Canada's Pesticide Managment Regulatory Agency (PMRA), and the California Department of Pesticide Regulation**. The handlers monitored include a treatment operator and treatment assistant. Handlers performed typical tasks related to these activities and were monitored during a full work cycle beginning at 7 AM and ending at 3 PM. The following are descriptions of the tasks:

- **Treatment operator** -TOs operated and monitored application system valves and controls, occasionally opened and closed cylinder doors, and supervised the insertion and removal of charges (loads of dried, debarked poles or untreated ties) of poles from the treatment cylinders.

- **Treatment assistant -**TAs performed many of the same functions as the TOs and sometimes assisted the TO during charge preparation, cylinder cleaning and maintenance, filter cleaning, mixing of treatment solution, and also participated in some of the loader operations for moving charges.

- **Loader operator** -LOs stacked untreated wood onto charge trams, moved charges into and out of treatment cylinders, distributed treated wood to load-out area, and loaded treated wood for shipment.

- **Cylinder-area helpers (CHs in the cylinder area, and LHs in the load-out areas)** -CHs/LHs aided the LOs by opening/closing cylinder doors, cleaning door debris and performing door maintenance, handling charge leads and cables, and banding stacked wood.

- **Checker (CK)** - CKs performed many of the duties of a CH.

- **Test borers (TBs)** - TBs took cores from freshly treated poles or ties to be tested for creosote content and penetration depth.

- **Load-out area helpers (LHs)** - LHs aided their LOs by banding treated wood and removing culls.

- **Oil unloaders (OUs)** - OUs operated the equipment that transferred creosote from rail tank cars to treating system tanks.

- **Drip pad laborer (DP)** - DPs steam-cleaned drip pads and tracks. They also picked up and disposed of treated wood waste and performed various labor clean-up duties in treatment areas.

- **Water treatment system operators (WOs)** - WOs controlled equipment that collected drip-pad effluent water, and removed creosote and other contaminants.

Pressure Treatment Process: Pressure treatment is often required because of the resistance of wood to deep penetration by preservatives. The pressure treatment process begins when untreated wood is loaded onto small rail/tram cars that are pushed into the treating cylinder using locomotives, forklifts, or similar equipment. The cylinder door is sealed via a pressure-tight door and the operation remains a closed system during the entire treatment process. Treating solutions are then pumped into the cylinder and the inside pressure is raised. At the end of the treatment process, the excess treating solution is pumped out of the treating cylinder and back to storage for reuse. The cylinder is opened, and the rail/tram cars holding the treated wood are pulled out of the cylinder using a locomotive, forklift, or similar equipment.

According to information provided by industry sources (Krygsman, 1994), wood pressure treatment of railroad ties in a retort may last anywhere from 4 to 24 hours. A typical retort cylinder has a diameter of about 8 feet and a length of about 120 feet. About 16 rail/tram cars can be placed in a retort at one time. The rail/tram cars usually are connected together and are pushed in and out of the retort on railroad tracks using a locomotive. Wood preservative is loaded into the wood pressure treatment retort facilities from rail tank cars using hoses and metered pumps. The wood preservative is stored in two or three holding tanks that may be as large as 60,000 gallons. During the wood treatment process the wood is sprayed under pressure in the enclosed retort. In the retort, a "charge" of liquid preservative is pumped into the trams and then later pumped out. After the wood preservative is pumped out, the wood is dried through a vacuum treatment and the tram cars containing wood (e.g. railroad ties) are then pulled out. Since the wood in the tram cars is pulled by mechanical means there is very little direct human contact with the exposed wood. Likely contact is through dermal contact with equipment that was previously in the retort, removing cables that separated layers of ties, dermal and inhalation contact to vapors inside the retort before and after pressure treatment, cleaning the retort, and inspecting wood pieces by coring the wood.

Dermal Exposure Study: Since creosote is a complex mixture of over 100 chemicals including phenols, creosol, and aromatic hydrocarbons, it is difficult and expensive to identify all of the chemicals in the mix. In addition, creosote cannot be measured directly because of its complex mixture. Dermal exposure to "total creosote" was estimated by measuring the levels of ten individual polynuclear aromatic hydrocarbon (PNA) compounds. Each analyte was determined in each whole-body dosimeter (WBD) and glove sample as if it represented total creosote. The goal was to use these marker compounds to represent "total creosote".

Dermal Exposure Monitoring: The creosote dermal exposure to each worker was determined using a WBD, consisting of a 100% cotton thermal shirt and long pants. Each worker at Sites A, C, or D wore his WBD under a fresh work uniform consisting of a cotton long-sleeved work shirt and cotton work trousers (or one-piece cotton coverall) provided by the test site. The workers at Site B were not provided uniforms therefore, for the purpose of this study, each worker wore a WBD under a fresh lightweight cotton/polyester sweat shirt and pants purchased locally by study personnel. The workers at all four sites wore a lightweight 100% cotton glove dosimeter on each hand under his chemical-resistant or work gloves, as appropriate. Each of these analytes were determined in each WBD and glove sample as if it represented total creosote. The averaged analyte concentrations were used to estimate the level of total creosote present in/on the individual sample.

Inhalation Exposure Study: Inhalation exposures for each worker was estimated by active dosimetry. Inhalation exposure was estimated for 11 individual PNA compounds as well as for benzene-soluble PNAs and related compounds collectively known as coal tar pitch volatiles (CTPVs). The Polytetrafluroethylene (PTFE) filter retained the CTPVs, while the PNAs were retained in the XAD-2 resin tubes. Each worker wore a sampling train consisting of a PTFE filter upstream from two in-line XAD-2 resin-filled air sampling tubes. (However, there was no attempt by the study sponsors to relate inhalation levels found for PNAs and CTPVs to "total creosote" -- a significant weakness with the study.)

Inhalation Exposure Monitoring: Handler inhalation exposure for a pressure treatment facility was examined using the creosote-specific data for the treatment operator and the treatment assistant.

Inhalation exposure monitoring at Site A was unsuccessful because a single XAD-2 tube was used along with a non-solvent-resistant filter cassette. Therefore, the sampling methodology was changed to include the use of a second XAD-2 resin tube in the sampling train prior to sampling at Sites B, C, and D. Inhalation exposure monitoring was performed successfully at these sites. Each worker at Sites B, C, and D was equipped with an air sampling train consisting of a PTFE filter in an opaque, solvent-resistant plastic cassette connected upstream from two in-line XAD-2 resin-filled air sampling tubes. The intake orifice of the filter was placed in the worker's breathing zone, directed downward. Air was pulled through the sampling train by a portable air sampling pump attached to the worker's belt. The pump drew air through the sampling tube at approximately 1 L/minute

while the worker performed his tasks. Pumps were calibrated immediately prior to and after each monitoring period using a mass flow meter or bubble calibrator. The pumps were turned on at the beginning of each work cycle and were left running during restroom, coffee, or other short breaks, but were turned off or set on "hold" during lunch breaks. The pumps and samplers were removed from the worker during the lunch break. At the conclusion of the lunch break, the pump and sampling train were reinstalled and the pump restarted. All start and stop times for breaks were recorded.

During each work cycle, start times and end times of each task performed by the worker were recorded. Pump parameters during use were also recorded. At the end of each work cycle, the pumps and sample trains were collected. Each filter cassette and sampling tube were capped, labeled, bagged, and placed on dry ice for shipment to USX Engineers and Consultants, Inc. (UEC) for extraction and analysis. After the collection of the air samples, the air sampling pump was re-calibrated.

4.2.2 Handler Risk Assessment and Characterization

The handler exposure assessment is based on the Creosote Council's worker exposure study. Tables 4 and 5 present the exposure/risk calculations for each exposure scenario.

The short-term dermal endpoint is based on a maternal toxicological endpoint; therefore, the body weight of a typical woman (60 kg) was used for the dose calculation. The adult body weight of 70 kg was used for the intermediate-term, long-term, and cancer endpoints. Short-term, long-term, and cancer endpoints are all based on oral administrations. A 50 percent absorption factor was used to develop a dermal dose based on an oral administration. Intermediate-term endpoints are based on a dermal administration; therefore, no absorption factor was used.

Creosote-specific inhalation data were available. For these scenarios, specific inhalation studies, which measured air concentrations in pressure treatment facilities, were used to derive an inhalation dose. The inhalation dose from an air concentration was calculated as follows:

$$Daily\ Inh.\ Dose\left(\frac{mg\ ai}{kg/day}\right) = Daily\ Exposure\ Data\ (mg/day)\ x\left(\frac{1}{Body\ Weight\ (kg)}\right)\ x\ ABS\ (\%)$$

Daily Exposure Data	=	Values obtained from studies
Body Weight (kg)	=	70 kg for short, intermediate and chronic
ABS	=	100 percent

Table 4. Handler Exposure/Dose for Creosote

Exposure Scenario[a]	Dermal						Inhalation			Combined Dermal & Inhalation Lifetime Average Daily Dose[g] (mg/kg/day)
	Exposure Study Data (μg/kg/day)[b]	Daily Exposure (mg/day)[c]	Short-term Daily Dose (mg/kg/day)[d]	Int.-term Daily Dose (mg/kg/day)[d]	Long-term Daily Dose (mg/kg/day)[d]	Lifetime Average Daily Dose (mg/kg/day)[e]	Daily Exposure[c] (mg/day)	Daily Dose[d] (mg/kg/day)	Lifetime Average Daily Dose[e] (mg/kg/day)	
Mixer/Loaders										
(1a) Mixing/Loading/ Applying Liquids at a Pressure Facility (treatment operator)	360	26	0.22	0.37	0.18	0.067	8.7[f]	0.12	0.045	0.11
(1b) Mixing/Loading/ Applying Liquids at a Pressure Treatment Facility (treatment assistant)	27	1.9	0.016	0.028	0.014	0.0051	4.9	0.07	0.026	0.031

a Exposure scenarios based on review of available labels and LUIS report.

b **Exposure Study Data (μg/kg/day): Exposure Study Data as provided in the study which was based on exposure to "total creosote" which was estimated by measuring the levels of ten individual polynuclear aromatic hydrocarbon (PNA) compounds. Each analyte was determined in each WBD and glove sample as if it represented total creosote. The goal was to use these marker compounds to represent "total creosote". The geometric mean of the ten marker compounds were taken for all sites and were used to represent total creosote exposure and normalized using , a body weight of 71.8 kg and a standard work day of 8 hours.**

Scenarios (1a,1b): Chemical specific exposure study data (Creosote Council II, 2001) for both a treatment operator and a treatment assistant submitted by the Creosote Council II. (Creosote Council II, 2001). Handlers wearing baseline attire and chemical resistant gloves.

c **Daily Exposure (mg/day): Exposure Study Data based on ten marker compounds taken to represent total creosote exposure.**

Scenarios (1a,1b): Chemical specific exposure study data (Creosote Council II, 2001) for both a treatment operator and a treatment assistant submitted by the Creosote Council II. (Creosote Council II, 2001). Handlers wearing baseline attire and chemical resistant gloves.

d Daily Dose (mg/kg/day) = Daily Dermal Exposure (mg/day) / Body Weight (kg). Short-term uses a 60 kg body weight and a 50% dermal absorption, intermediate-term uses a 70 kg body weight with no absorption, and long-term uses a 70 kg body weight with 50% dermal absorption. Inhalation uses a 70 kg body weight with 100% absorption.

e Lifetime Average Daily Creosote Dose (mg/kg/day)=Daily Exposure (mg/day) / Body Weight (70 kg) * ABS (50%) * [Exposure Frequency (250 days/year) * Exposure Duration (40 yrs)] / [365 days/yr * Lifetime (75 yrs)].

f Based on the study submitted by Creosote Council II entitled" Assessment of Potential Creosote Inhalation and Dermal Exposure Associated with Pressure-Treatment of Wood with Creosote" (Creosote Council II, 2001). Note: Inhalation exposure was estimated for 11 individual PNA compounds as well as for benzene-soluble PNAs and related compounds collectively known as coal tar pitch volatiles (CTPVs).

g Combined dermal and inhalation lifetime average daily dose (LADD) = dermal LADD + inhalation LADD.

NA - Not available.

GM - Geometric mean. The study provided the geometric mean creosote exposure based on multiple replicates from multiple sites presented in the study submitted by Creosote Council II (2001). The geometric mean exposure is used to represent a typical exposure.

Table 5. Handler Short-term, Intermediate-term, and Long-term Risks for Creosote

Exposure Scenario[a]	Risk Mitigation [b]	Dermal MOEs			Inhalation MOE [f]	Cancer Risks Based on 0.5-1.0% Benzo[a] Pyrene[g]
		Short-term [c]	Intermediate-term [d]	Long-term [e]		
Mixer/Loaders						
(1a) Mixing/Loading/Applying Liquids at a Pressure Treatment Facility (treatment operator)	closed mixing	230	110	140	9.7	4.1E-03-8.2E-03
(1b) Mixing/Loading/Applying Liquids at a Pressure Treatment Facility (treatment assistant)	closed mixing	3100	1400	1800	17	1.1E-03-2.2E-03

[a] Exposure scenarios based on review of available labels and LUIS report.

[b] Risk mitigation is per each facility in the pressure treatment exposure study.

[c] Short-term Dermal MOE= Short-term NOAEL (50 mg/kg/day) / Short-term Dose (see Table 4).

[d] Intermediate-term Dermal MOE= Intermediate-term NOAEL (40 mg/kg/day) / Intermediate-term Dose (see Table 4).

[e] Long-term Dermal MOE= Long-term LOAEL (25 mg/kg/day)/ Long-term Dose (see Table 4).

[f] Inhalation MOE= Inhalation NOAEL (1.2 mg/kg/day)/ Inhalation Dose (see Table 4).

[g] Cancer Risk= Combined Lifetime Average Daily Dose (LADD) (see Table 4) x Q_1 * (7.3 $(mg/kg/day)^{-1}$) x (0.5% or 1.0% benzo[a]Pyrene).

Creosote-specific dermal data were available for scenarios 1a and 1b. The dermal dose was calculated as follows:

$$Daily\ Dermal\ Dose\left(\frac{mg\ ai}{Kg/Day}\right) = Daily\ Dermal\ Exposure\left(\frac{mg\ ai}{day}\right) x \left(\frac{1}{Body\ Weight\ (Kg)}\right) x\ ABS\ (\%)$$

Daily Dermal Exposure	=	Exposure study data (ug/kg/day)x Study's Body Weight (71.8 kg)
Body Weight (kg)	=	60 kg for short-term and 70 kg for intermediate-term and chronic

Table 4 uses exposure study data from the Creosote Council II, 2001, exposure assessment entitled "Assessment of Potential Creosote Inhalation and Dermal Exposure Associated With Pressure-Treatment of Wood with Creosote." This source provides a geometric mean dermal dose of 360 µg/kg/day (note: the maximum dermal dose is 49,573 µg/kg/day) for a treatment operator (scenario 1a, Table 4) and a geometric mean dermal dose of 27 µg/kg/day (note:the maximum dermal dose is 33 µg/kg/day) for a treatment assistant (scenario 1b, Table 4). Because EPA traditionally uses an adult body weight of 70 kg and female body weight of 60 kg in its exposure assessments which is slightly different then the 71.8 kg body weight used in the Creosote Council II exposure assessment, the doses used in this assessment had to be normalized back to daily dermal exposures. The normalization was performed by multiplying the exposure dose times the 71.8 kg body weight.

ABS	=	50 percent absorption for short-term and chronic, 100 percent for intermediate-term

The calculations of the daily dermal dose of creosote received by handlers were used to calculate the short-term, intermediate-term, and chronic MOEs. The daily dermal MOE was calculated using an NOAEL of 50 mg/kg/day for short-term exposure, an NOAEL of 40 mg/kg/day for intermediate-term exposures, and an LOAEL of 25 mg/kg/day for chronic exposures.

Risk Calculations: The following formula describes the calculation of a dermal MOE:

$$Dermal\ MOE = \left(\frac{NOAEL\ (mg/kg/day)}{Dermal\ Dose\ (mg/kg/day)} \right)$$

The target MOE for short- and intermediate-term dermal exposure is 100 and the target MOE for long term exposure is 300.

The following formula describes the calculation of an inhalation MOE:

$$Inhalation\ MOE = \left(\frac{NOAEL\ (mg/kg/day)}{Inhalation\ Dose\ (mg/kg/day)} \right)$$

The target MOE for total short-term, intermediate-term, and long-term inhalation exposure is 100.

Handler Exposure and Cancer Risk Calculations: For the handler exposure and cancer risk calculations the lifetime average daily dose was calculated by adding the chronic dermal and inhalation doses and accounting for exposure frequency, exposure duration, and lifetime. Exposure duration was assumed to be 40 years and is the standard value used by EPA Office of Pesticide Programs to represent a working lifetime. This is assumed to be a high end value. Lifetime is assumed to be 75 years. This is the recommended value for the U.S. population as cited in EPA's Exposure Factors Handbook (USEPA, 1997). All handler scenarios assume an exposure frequency of 250 days per year (i.e., 5 days per week, 50 weeks per year). This is a standard Agency assumption for days worked per year. Table 5 details the handler cancer risk estimates. The following formula describes the calculation of the lifetime average daily dose (LADD):

$$LADD\ (mg/kg/day) = \frac{Daily\ Exp.\ (mg/day)/Body\ Weight\ (kg) * ABS\ (\%) * Exp.\ Frequency\ (days) * Exp.\ Duration\ (yrs)}{365\ days/yr * Lifetime\ (yrs)}$$

Risks are calculated by multiplying the lifetime average daily dose times the cancer slope factor of 7.3 $(mg/kg/day)^{-1}$ using the following formula:

$$Risk = LADD \left(\frac{mg\ ai}{kg/day} \right) \times Cancer\ Slope\ Factor \left(\frac{1}{(mg/kg/day)} \right)$$

Creosote is rated as a B1 probable human carcinogen based on limited evidence of the association between occupational creosote contact and subsequent tumor formation. Further, while a specific quantitative risk assessment on carcinogenicity of creosote has not been performed by the Agency, a quantitative cancer risk assessment exists for benzo(a)pyrene, one of the components of creosote. Administration of benzo(a)pyrene by inhalation has been shown to result in respiratory tract tumors, and administration by the dermal route results in skin tumor production, similar to creosote. Benzo(a)pyrene has also been shown to be a "complete" carcinogen similar to creosote, and also tests positive for mutagenicity on a variety of assays. Therefore, the Anitmicrobials Division is using the cancer slope factor for benzo(a)pyrene [7.3 $(mg/kg/day)^{-1}$] as an indicator of worker risk in conducting the cancer risk assessment for creosote.

Non-cancer acute, sub-chronic, and chronic toxicity endpoints related to dermal exposures to creosote have also been identified. A MOE of greater than 100 for creosote is considered to indicate no risk concern for short-term and intermediate-term exposures, and a MOE of greater than 300 for creosote is considered to indicate no risk concern for chronic exposures. The results were presented in Table 5 and are summarized as follows:

The calculations of short- and intermediate-term risks indicate that dermal MOEs are more than 100 (i.e., not of concern) with additional engineering controls for the following scenarios:

- (1a) **Mixing/loading/applying liquids at a pressure treatment facility - treatment operator.**
- (1b) **Mixing/loading/applying liquids at a pressure treatment facility - treatment assistant.**

The calculations of long-term risks indicate that dermal MOEs are less than 300 with additional engineering controls for the following scenarios:

- (1a) **Mixing/loading/applying liquids at a pressure treatment facility - treatment operator**.

The calculations of long-term risks indicate that dermal MOEs are more than 300 with additional engineering controls for the following scenarios:

- (1b) **Mixing/loading/applying liquids at a pressure treatment facility - treatment assistant**.

One inhalation endpoint (acute, sub-chronic, and chronic) related to inhalation exposures has been identified. A MOE of greater than 100 for creosote is considered to indicate a no-risk exposure. The creosote inhalation MOEs for the TO and TA are of concern (MOEs = 10 and 17, respectively).

All of the handler scenarios exceed the 1E-04 cancer risk levels. All of the handler scenarios are expected to pose a risk concern.

4.3 Occupational Post-application Exposures and Risks

The Agency is concerned about potential post-application exposures to creosote. Since coal tar creosote is a blend of over 100 compounds, degradation is complicated. These compounds include volatile and semi-volatiles. The volatiles are the single ring compounds and the semi-volatiles are the two to six ring compounds. The vapor pressure tends to become larger as aromatic rings are added to the compound. The more soluble compounds in creosote include phenols, creosol, and N-heterocyclics. The high molecular weight PAHs tend to have low aqueous solubilities ("The Environmental Degradation of Creosote", 1998).

Potential post-application exposure may occur following creosote applications in commercial, industrial, and residential settings. Post-application concerns exist in residential settings when pressurized treated wood (railroad crossties, cross planks, cross arms) is used for

block flooring, and fence posts in residential areas. Although homeowner handler use is prohibited by the label, post-application exposures to creosote-treated wood are a potential homeowner concern.

The potential post-application occupational exposures include:

(1) cylinder area loader operator;
(2) cylinder area loader helper;
(3) checker;
(4) drip pad laborer;
(5) load out area loader operator;
(6) load out area loader helper;
(7) load out area forklift operator;
(8) oil unloader;
(9) pressure treatment test borer;
(10) water treatment systems operator;
(11) railroad installer; and,
(12) pole installer.

The potential post-application exposures to homeowners include:

(1) homeowner incidental ingestion and dermal contact with soil contaminated with creosote (e.g., soil contaminated by creosote treated telephone poles) (child)
(2) outdoor homeowner dermal contact with industry pressure treated wood products (e.g., utility poles, piers, etc.) (adult)
(3) outdoor homeowner incidental hand-to-mouth and dermal contact with industry pressure treated wood products (e.g., utility poles, posts, decks, shingles, fencing, lumber, piers, etc.) (child)

4.3.1 Post-application Occupational Data and Assumptions, and Exposure and Risk Calculations

In the course of development of this RED, chemical-specific post-application data identified from pertinent literature sources were used in conjunction with both industry and Agency estimates of exposure parameters to predict exposures. Tables 6 and 7 include the exposure/risk calculations for non-cancer and cancer risks for each exposure scenario.

Table 6. Post-application Exposure/Dose for Creosote

Exposure Scenario[a]	Dermal						Inhalation			Combined Dermal & Inhalation Lifetime Average Daily Dose [f] (mg/kg/day)
	Exposure Study Data [b] (μg/kg/day)	Daily Exposure [c] (mg/day)	Short-term Daily Dose[d] (mg/kg/day)	Int.-term Daily Dose[d] (mg/kg/day)	Long-term Daily Dose[d] (mg/kg/day)	Lifetime Average Daily Dermal Dose [e] (mg/kg/day)	Daily Exposure [c] (mg/day)	Daily Dose[d] (mg/kg/day)	Lifetime Average Daily Dose [e] (mg/kg/day)	
(1) Cylinder Area Loader Operator	313	22	0.19	0.32	0.16	0.059	11	0.16	0.057	0.12
(2) Cylinder Area Loader Helper	626	45	0.37	0.64	0.32	0.12	22	0.31	0.11	0.23
(3) Checker	638	46	0.38	0.65	0.33	0.12	3.1	0.044	0.016	0.14
(4)Drip Pad Labor	271	19	0.16	0.28	0.14	0.051	4.4	0.063	0.023	0.074
(5) Load-out Area Loader Operator	69	5	0.041	0.071	0.035	0.013	4.4	0.063	0.023	0.036
(6) Load-out Area Loader Helper	25	1.8	0.015	0.026	0.013	0.0047	1.3	0.019	0.007	0.011
(7) Load-out Area Forklift Operator	208	15	0.12	0.21	0.11	0.039	8.3	0.12	0.043	0.082
(8) Oil Unloader	901	65	0.54	0.92	0.46	0.17	13	0.19	0.068	0.24
(9) Test Borer	385	28	0.23	0.39	0.2	0.072	11	0.16	0.057	0.13
(10) Water Treatment System Operator	108	7.8	0.065	0.11	0.055	0.02	7.7	0.11	0.040	0.06
(11) Railroad Worker	No Data	No Data	No Data	No Data	No Data	No Data	No Data	No Data	No Data	No Data
(12) Pole Installer	No Data	No Data	No Data	No Data	No Data	No Data	No Data	No Data	No Data	No Data

a Exposure scenarios based on review of available labels and LUIS report.

b Exposure Study Data - Based on the study submitted by Creosote Council II entitled "Assessment of Potential Creosote Inhalation and Dermal Exposure Associated with Pressure-Treatment of Wood with Creosote" (Creosote Council II, 2001). Daily Exposure (μg/kg/day): Exposure Study Data based on exposure to "total creosote" which was estimated by measuring the levels of ten individual polynuclear aromatic hydrocarbon (PNA) compounds. Each analyte was determined in each WBD and glove sample as if it represented total creosote. The ten marker compounds were averaged together to represent total creosote exposure and normalized using a body weight of 71.8 kg and a standard work day of 8 hours. Inhalation exposure results based on the study submitted by Creosote Council II entitled "Assessment of Potential Creosote Inhalation and Dermal Exposure Associated with Pressure-Treatment of Wood with Creosote" (Creosote Council II, 2001). Note: Inhalation exposure was estimated for 11 individual PNA compounds as well as for benzene-soluble PNAs and related compounds collectively known as coal tar pitch volatiles (CTPVs).

c Daily exposure = Exposure Study Data based on 11 individual PNA compounds to represent total creosote exposure. Data in units of µg/kg/day were multiplied by a body weight of 71.8 kg to convert to mg/day.

d Daily dose (mg/kg/day) = daily exposure / body weight (kg). Short-term uses a 60 kg body weight and a 50 percent dermal absorption; intermediate-term uses a 70 kg body weight (no absorption factor is necessary); and long-term uses a 70 kg body weight with 50 percent dermal absorption. Inhalation uses a 70 kg body weight with 100 percent inhalation absorption.

e Lifetime average daily dose = daily dose (mg/kg/day) * [exposure frequency (250 days/year) * exposure duration (40 years)] / [365 days/year * lifetime (75 years)].

f Combined LADD = dermal LADD + inhalation LADD.

Table 7. Post-application Short-term, Intermediate-term, and Long-term Risks for Creosote

Exposure Scenario[a]	Dermal MOEs			Inhalation MOE [e] Target MOE=100	Cancer Risk based on 0.5%-1.0% Benzo [a] Pyrene[f]
	Short-term [b] Target MOE=100	Intermediate-term [c] Target MOE=100	Long-term [d] Target MOE=300		
(1) Cylinder Area Loader Operator	270	120	160	7.6	4.3E-03-8.5E-03
(2) Cylinder Area Loader Helper	130	62	78	3.8	8.5E-03-1.7E-02
(3) Checker	130	61	76	27	5.0E-03-9.9E-03
(4)Drip Pad Labor	310	140	180	19	2.7E-03-5.4E-03
(5) Load-out Area Loader Operator	1200	570	710	19	1.3E-03-2.6E-03
(6) Load-out Area Loader Helper	3300	1600	1900	65	4.2E-04-8.4E-04
(7) Load-out Area Forklift Operator	400	190	230	10	3.0E-03-6.0E-03
(8) Oil Unloader	93	43	54	6.5	8.5E-03-1.7E-02
(9) Test Borer	220	100	130	7.6	4.7E-03-9.4E-03
(10) Water Treatment System Operator	770	360	450	11	2.2E-03-4.4E-03
(11) Railroad Worker	No Data	No Data	No Data	No Data	No Data
(12) Pole Installer	No Data	No Data	No Data	No Data	No Data

[a] Exposure scenarios based on review of available labels and LUIS report.

[b] Short-term Dermal MOE= Short-term NOAEL (50 mg/kg/day) / Short-term Dose (see Table 6).

[c] Intermediate-term Dermal MOE= Intermediate-term NOAEL (40 mg/kg/day) / Intermediate-term Dose (see Table 6).

[d] Long-term Dermal MOE= Long-term LOAEL (25 mg/kg/day)/ Long-term Dose (see Table 6).

[e] Inhalation MOE= Inhalation NOAEL (1.2 mg/kg/day)/ Chronic Dose (see Table 6).

f Cancer risk = combined LADD (see Table 6) * cancer slope factor $(7.3\ mg/kg/day)^{-1}$ x (0.5% or 1.0% Benzo[a]Pyrene).

Dermal Exposure Studies: The Creosote Council II exposure study data that were used for handler exposure were also used for post-application dermal exposure (Creosote Council II, 2001). No other data for dermal post-application exposure were identified in the available literature. The difference between the reported data for the handler and the post-application assessment was that the exposure data for the treatment operator and treatment assistant were used as representative of handler exposures, and the exposure data for cylinder area loader, cylinder area loader helper, checker, drip pad loader, load-out area loader, load-out area loader helper, load-out area forklift operator, oil unloaders, test borers, water treatment system operator, railroad worker and pole installer were assumed to be representative of dermal post-application exposure to creosote at a pressure treatment facility. The individuals are representative of post-application exposures because they were exposed to pressure treated wood following treatment with creosote. The methods and deficiencies of the dermal exposure study as well as a brief description of the pressure treatment process were fully described in Section 4.2.a. A description of the scenarios are listed below:

1) **Loader operators (CLOs in the cylinder area, and LLOs in the load-out areas)** - LOs stacked untreated wood onto charge trams, moved charges into and out of treatment cylinders, distributed treated wood to load-out area, and loaded treated wood for shipment.

2) **Cylinder-area helpers (CHs in the cylinder area, and LHs in the load-out areas)** - CHs/LHs aided the LOs by opening/closing cylinder door, cleaning door debris and performing door maintenance, handling charge leads and cables, and banding stacked wood.

3) **Checker (CK)** - CKs performed many of the duties of a CH.

4) **Load-out area helpers (LHs)** - LHs aided their LOs by banding treated wood and removing culls.

5) **Test borers (TBs)** - TBs took cores from freshly treated poles or ties to be tested for creosote content and penetration depth.

6) **Oil unloaders (OUs)** - OUs operated the equipment that transferred creosote from rail tank cars to treating system tanks.

7) **Drip pad laborer (DP)** - DPs steam-cleaned drip pads and tracks. They also picked up and disposed of treated wood waste and performed various labor clean-up duties in treatment areas.

8) **Water treatment system operators (WOs)** - WOs controlled equipment that collected drip-pad effluent water, and removed creosote and other contaminants.

9) **Railroad worker**- This individual is assumed to become exposed during the mechanical and manual installation of pressure treated railroad crossties as well as during inspection procedures (ATSDR, 1990). No dermal exposure data were available for this scenario.

10) **Pole installers-** This individual was expected to become exposed while attaching fittings on telephone poles, installing new telephone poles, conducting groundline treatment of telephone poles, and maintaining and repairing existing telephone poles (ATSDR, 1990). No dermal exposure data were available for this scenario.

Chemical-specific data were available for all scenarios except for the Railroad worker and Pole installer scenarios. The dermal dose was calculated as follows:

$$Daily\ Dermal\ Dose\left(\frac{mg\ ai}{Kg/Day}\right) = Daily\ Dermal\ Exposure\left(\frac{mg\ ai}{day}\right) x \left(\frac{1}{Body\ Weight\ (Kg)}\right) x\ ABS\ (\%)$$

Daily Dermal Exposure = Exposure Study data (ug/kg/day) x Study Body Weight (71.8 kg).

The column entitled Exposure Study Data in Table 6 presents the dermal doses calculated by the Creosote Council II, 1998 in an exposure assessment entitled "Assessment of Potential Creosote Inhalation and Dermal Exposure Associated With Pressure-Treatment of Wood with Creosote". Because EPA traditionally uses an adult body weight of 70 kg and female body weight of 60 kg in its exposure assessments which is slightly different then the 71.8 kg body weight used in the Creosote Council II exposure assessment, the doses used in this assessment had to be normalized back to daily dermal exposures. The normalization was performed by multiplying the exposure dose times the 71.8 kg body weight

Body Weight (kg)	=	60 kg for short-term, 70 kg for intermediate-term and chronic
ABS	=	50 percent absorption for short-term and chronic; 100 percent absorption for intermediate-term

The calculations of the daily dermal dose of creosote received by handlers were used to calculate the short-term, intermediate-term, and chronic MOEs.

Inhalation Exposure Studies: The scenarios for inhalation post-application exposure are identical to that of the dermal data. The data from the Creosote Council II were used for this assessment and used to estimate inhalation doses.

Creosote inhalation dose was calculated from an air concentration as follows:

$$Daily\ Inh.\ Creosote\ Dose\left(\frac{mg\ ai}{kg/day}\right) = Daily\ Inhalation\ Creosote\ Exposure\left(\frac{mg}{day}\right) x \left(\frac{1}{Body\ Weight\ (kg)}\right)$$

Daily exposure inhalation creosote	=	mg/day
Body Weight (kg)	=	70 kg for short- and intermediate-term and chronic exposure

The daily inhalation MOE was calculated using a NOAEL of 1.2 mg/kg/day.

Summary of Other Exposure Studies: Additional creosote exposure studies in the literature are summarized below and in Table 8. Todd and Timbie (NIOSH; 1980) estimated occupational exposures of workers to creosote in a railroad tie treatment plant in Sommerville, Texas. Petroleum oil/creosote solutions of 70/30 and 50/50 were used respectively to treat the cross ties and bridge timbers in the plant. The concentrations of creosote (i.e., coal-tar pitch volatiles; CTPV) in personal air samples over a two-day monitoring period ranged from 0.002 to 1.211 mg/m^3.

Table 8. Summary of Occupational Inhalation Exposure Studies of Creosote

Study	Setting/Subjects	Components Reported (Analyzed)	Concentration
NIOSH; 1980 (Todd & Timbie)	railroad tie treatment plant	coal-tar pitch volatiles (CTPV)	0.002-1.211 mg/m^3
NIOSH; 1981a (Todd & Timbie)	wood treatment facility	CTPV	0.0004-0.112 mg/m^3
NIOSH; 1981b (Baker &Fannick)	dock builder	CTPV (cyclohexane extractables)	0-0.059 mg/m^3
Markel et al. (1977) and SRI (1993)	wood treatment facility	polycyclic organic materials (PPOM)	<0.1 mg/m^3
Hiekkila et al. (1987)	creosote impregnation plant;	average total vapor (naphthalene being the major component)	0.5-71 mg/m^3
Hiekkila et al. (1987)	handling impregnated wood	average total vapor (naphthalene being the major component)	0.1-11mg/m^3
Flickenger and Lawrence, 1982	wood pressure treatment plants	total vapor (naphthalene being the major component)	0.92-6.5 mg/m^3

Another NIOSH study of occupational exposure to creosote at a wood-treatment facility in Tacoma, Washington reported CTPV concentrations in personal air samples ranging from less than 0.0004 to 0.112 mg/m^3 with the highest concentration found at the end of the treatment process when the cylinder was opened (NIOSH; 1981a). NIOSH also reported creosote exposures of dock builders ranging from zero to 0.059 mg/m^3 based on cyclohexane extractable fraction of CTPV (NIOSH; 1981b).

Studies conducted by Markel et al. (1977) and SRI (1993) indicated that particulate polycyclic organic materials (PPOM) was within 0.1 mg/m^3, the NIOSH permissible level for CTPV, when estimating occupational exposure to creosote in wood

treatment plants. The concentrations of naphthalene, methylnaphthalene, and acenaphthene (the only components in the vapor-phase fractions that could be reliably measured) ranged from 0.54 to 2.0 mg/m^3. Benzene-soluble particulates (PPOM) ranged from 0.02 to 0.10 mg/m^3.

Hiekkila et al. (1987) conducted an occupational study in Finland estimating workers' exposure to creosote in the creosote impregnation plants and when they were handling the impregnated wood. The average vapor concentrations (naphthalene being the major component) ranged from 0.5 to 71 mg/m^3 in the impregnation plants; while the vapor concentrations ranged from 0.1 to 11 mg/m^3 in the handling of impregnated wood. Most of the airborne contaminants in workers' breathing zones were in the vapor phase; the proportion of particulate polycyclic aromatic hydrocarbons (PAHs) to total concentration of vapors was less than 0.5 to 3.7 percent.

A German study by Rotard and Mailahn (1987) reported high levels of carcinogenic PAHs, such as benzo[a]pyrene, benzo[b]-fluoranthene, and benzo[j]fluoranthene, and cocarcinogenic PAHs in samples of wooden sleepers (railroad cross ties) installed in playgrounds.

A study entitled "Occupational Health Experience in the Wood Preserving Industry" MRID 447595-02 was submitted to the U.S. Environmental Protection Agency (EPA) by industry for review as a possible source of handler and post-application inhalation information for the Creosote Reregistration Eligibility Decision Document (RED) exposure chapter (Flickinger and Lawrence, 1982). This study assessed the daily inhalation exposure of workers involved in the wood pressure treatment industry. The requirements for inhalation exposure are normally specified by the U.S. Environmental Protection Agency under *Series 875.1300 Group A Occupational and Residential Exposure Test Guidelines and Series 875.2500 Group B - Post Application Exposure Monitoring Test Guidelines.* The EPA reviewed this study and concluded that the study does not meet the minimum Series 875 guidelines and has several issues of technical merit. The major issues of technical merit are listed below:

- A thorough description of the inhalation sampling equipment (e.g., type of personal sampling pumps and attached collection devices), sampling technique

(e.g, duration of time the samples were collected), equipment calibration, air flows, EPA sampling method numbers were not provided.

- The detectable concentrations have a wide range of variability.

- The study appeared to be too old (e.g., the study was written in 1982 and the sampling analysis results were performed in 1977) to represent current conditions at a creosote pressure treatment facility and does not begin to address all of the issues presented in the Series 875 guidelines.
- The study does not assess dermal exposure.

- The study does not specify whether the creosote was a P2 or P1/P13 formulation.

- Background creosote air concentrations were not provided.

- Limits of detection and quantification limits were not clearly identified.

- Personal protection methods and engineering controls were apparently used, but not described.

The following is a brief description of the Series 875 compliance issues:

- Series 875 requires tests on laboratory recovery, field recovery, or method recovery to evaluate the overall quality of the analysis. A study must have a recovery percent of between 70-120% with a standard deviation of one to be acceptable. Since the study does not discuss recovery, it is impossible to determine extraction and analysis efficiency. If the recovery is not acceptable, the data must be corrected to determine the actual concentrations.

- The study does not discuss storage stability issues. Storage stability issues discuss how long between collection and analysis and possible degradation of the compound in question.

- Series 875 requires that the study identify the"typical end use product of the active ingredient used." The study identifies the end use product as Koppers 70/30 creosote/coal tar solution; however, the study does not reference a label, provide an analysis of the batch, or identify typical uses.

- It was not clear how many sites were evaluated. Series 875 guidelines recommend that at least three representative sites should be selected.

- Series 875 guidelines require that "for exposure monitoring at least five replicates (e.g., individuals) at each of three monitoring periods be assessed (e.g., 'n' days after application)." The replicates of the workers were not reported and all workers were monitored on just one day.

- Series 875 guidelines recommend that "monitoring period is sufficient to collect measurable residues, but not excessive so that residue loss occurs." Sampling duration was not discussed.

- Series 875 guidelines requires that "inhalation exposures be monitored by validated methodologies". Methods were briefly discussed, but no specified method numbers were referenced.

- Series 875 guidelines requires that "quantity of active ingredient handled and duration of monitoring period should be reported for each replication." This was not reported.

- Series 875 guidelines require that quantitation of level of detection be reported.

- Series 875 guidelines require that "at least one field fortification sample per worker per monitoring period per fortification level for each matrix. At least one field blank per worker per monitoring period for each matrix." No field blanks or fortification samples were reported.

This study was rejected in favor of the more recent Creosote Council II study (Creosote Council II, 2001). The major reasons include: (1) the study would better represent more current conditions at wood preservative plant; (2) the study included an

analysis of field, lab and method recoveries; (3) the pilot study also assessed dermal exposure; (4) the study addressed important Series 875 issues such as number of replicates, sampling methodologies, fortifications, and levels of detection and quantitation; and (5) the Creosote Council II study is more recent.

Post-application Cancer Risk Calculations: Post-application cancer risks were calculated in the same manner as for handlers. The exposure durations and lifetime values used were the same as for handlers. Exposure frequency was assumed to be 250 days/year (i.e., standard annual working frequency) for all scenarios. Estimated cancer risks from dermal and inhalation post-application exposures are presented in Table 8.

4.3.2 Occupational Post-application Risk Assessment and Characterization

The calculations of short-term dermal non-cancer risks indicate that dermal MOEs are more than 100 (i.e., not of concern)for the following scenarios:

- (1) Cylinder area loader operator
- (2) Cylinder area loader helper
- (3) Checker
- (4) Drip pad labor
- (5) Load-out area loader operator
- (6) Load-out area loader helper
- (7) Load-out area forklift operator
- (9) Test borer
- (10) Water treatment system operator

Note: Scenario 8, oil unloader, the MOE is 93.

The calculations of intermediate-term risks indicate that dermal MOEs are more than 100 (i.e., not of concern) for the following scenarios:

- (1) Cylinder area loader operator
- (4) Drip pad labor
- (5) Load-out area loader operator
- (6) Load-out area loader helper
- (7) Load-out area forklift operator
- (9) Test borer (MOE is 100)
- (10) Water treatment system operator

The calculations of long-term risks indicate that dermal MOEs are more than 300 (not of concern) for the following scenarios:

- (5) Load-out area loader operator
- (6) Load-out area loader helper
- (10) Water treatment system operator

The calculations of short-, intermediate-, and long-term inhalation non-cancer risks indicate that inhalation MOEs are more than 100 for the following scenarios:

- None

For dermal and inhibitor cancer risks all of the post-application scenarios (except for scenarios 11 and 12 - see below) exceed the 1E-04 risk levels. All of these scenarios are expected to pose a risk concern.

Data gaps exist for the following scenarios:

- (11) Railroad workers
- (12) Pole installers

The Agency also has is concerns that there are potential exposure concerns relating to post-application exposure to individuals following the use of creosote-treated wood in residential settings. The potential residential post-application exposure pathways are outlined below:

(1) homeowner incidental ingestion and dermal contact with soil contaminated with creosote (e.g., soil contaminated by creosote treated telephone poles) (child)

(2) outdoor homeowner dermal contact with industry pressure treated wood products (e.g., utility poles, railroad ties used in home settings, posts) (adult)

(3) outdoor homeowner hand-to-mouth and dermal contact with industry pressure treated wood products (e.g., utility poles, railroad ties used in home settings, posts) (child)

No chemical-specific data for residential post-application exposure was submitted. Therefore, exposure doses could not be calculated. Data were not adequate for use in the exposure assessment.

4.4 Uncertainties and Limitations

4.4.1 Data Gaps

At this time, information from creosote labels, from EPA's LUIS database, and from industry sources has been used to identify probable use scenarios for creosote. These may have to be adjusted if more specific use information is received from industry sources.

Dermal and inhalation data gaps exist for the following post-application exposure scenarios:

- Railroad Worker, and
- Pole Installer.

Data are not adequate to characterize residential post-application exposure.

4.4.2 Creosote Council II 2001 Worker Exposure Study

This section summarizes key compliance concerns with using the Creosote Council II worker exposure study (Creosote Council II, 2001) which replaces the data from the pilot study (Creosote Council II, 1998). The Agency used a worker exposure study submitted from the Creosote Council II to provide chemical specific handler and exposure data post-application in support of re-registration of creosote (Creosote Council II, 2001). The Creosote Council II study was judged to be of better quality than the other currently existing exposure studies available at this time (NIOSH, 1980; NIOSH 1981a; NIOSH 1981b; Markel et al., 1977; SRI, 1993; Heikkila et al., 1987; and Flickenger and Lawrence, 1982). Compliance concerns for the Creosote Council (2001) study are the following:

- It should be noted that the Creosote Council II (2001) exposure study data were used for dermal and inhalation handler exposure and also for post-application dermal and inhalation exposure. The difference between the reported data for the handler and the post-application assessment was that the exposure data for the treatment operator and treatment assistant were used as representative of handler exposures, and the exposure data for cylinder area loader, cylinder area loader helper, checker, load-out area loader, load-out area loader helper, load-out area forklift operator, oil unloader, test borer, water treatment system operator, railroad worker, and pole installer were assumed to be representative of post-application exposure to creosote at a pressure treatment facility. These individuals are representative of post-application exposures because they were exposed to pressure treated wood following treatment with creosote.

- The amount of product applied and the amount of active ingredient handled by each worker was not calculated because the creosote was applied in a closed system which recovered and retained excess treatment solution from the wood and treatment vessel while sealed.

- The number of field fortification samples collected at the sites were less than the required number to satisfy Series 875 guidelines. According to the guidelines, there should be at least one fortification sample per worker per monitoring period (8 hour shift) per fortification level (three levels) for each

matrix and at least one field blank per worker per monitoring period for each matrix. There were more workers monitored than there were field fortifications and field blank samples collected.

- The overall inhalation field fortification percent recoveries for the coal tar pitch volatiles (CTPVS) were poor. The overall recovery for Site B was 57%. The overall recoveries for Sites C and D were 51% and 57%, respectively. All inhalation fortification recoveries below 70% should be considered unacceptable according to Series 875 guidelines and therefore undermines the validity of the results.

- There were some dermal fortification levels with extremely high recoveries for WBD's and some with unacceptable low recoveries for gloves. As an example, for a 60 µg/sample "total creosote" fortification for Site B, the recoveries for the WBD's were as high as 150% and recoveries for the gloves as low as 52.3%. There were measurable amounts of total creosote found in each of the control samples prepared at each facility. All dermal fortification recoveries above 120% and below 70% are outside of the range recommended in Series 875 guidelines and undermine the validity of the results.

- The study sponsors made no attempt to relate inhalation levels found for PNAs and CTPVs to "total creosote" -- a significant weakness with the study.

- PMRA has indicated that there are calculation mistakes with inhalation data in the study.

- There were inconsistencies in raw data and examples provided by the study authors: e.g., inhalation raw data did not reflect data found in bar graphs.

4.4.3 Residential Exposure Scenarios

No chemical-specific data for residential post-application exposure were submitted and exposures were not calculated. No chemical-specific data for residential exposure were identified in the available literature.

4.4.4 Toxicity Information

Cancer information was not initially presented in the initial draft of the Hazard Identification report (USEPA, 1999). Toxicity experts at the EPA recommended using the BAP cancer slope factor of 7.3 $(mg/kg/day)^{-1}$ as an indicator of worker risk concerns for creosote. This recommendation was based on the Agency's decision to use benzo(a)pyrene, a component of creosote, as a surrogate for identifying potential worker cancer risk concerns for creosote. Also, it is noted that creosote is classified as a B1 carcinogen, whereas benzo(a)pyrene is classified as a B2 carcinogen.

As indicated above, the Agency uses the risk assessment for benzo(a)pyrene as an indicator of worker risks for creosote. Considering this, the Agency adjusted calculated creosote handler and post-application cancer risk calculations by factors of 0.005 and 0.01. This was done because: (a) benzo(a)pyrene is a component found in creosote formulations; and (b) available information indicates that benzo(a)pyrene occurs as a component in creosote at levels of 0.5%. (However, in order to provide a conservative assessment the Agency assumed that levels of benzo(a)pyrene may occur from 0.5% to 1% of total creosote formulations.)

It should also be noted that although these corrections to cancer risk estimates were made, data from the Creosote Council II's 2001 worker exposure study were not provided on the actual amount of benzo(a)pyrene found as dermal residues. Further, in this study all inhalation samples of benzo(a)pyrene were found to be at levels below the Level of Detection (LOD). These factors, therefore, increase the uncertainty of the cancer risk assessment.

4.5 Results and Conclusions

The results of the handler exposure and risk assessment indicate that the risk drivers are the long-term dermal MOEs, inhalation MOEs, and the cancer assessment with the cancer risks of most concern. Cancer risks for all handler scenarios exceed the level of concern (1E-04) for occupational handlers. Table 9 summarizes each exposure pathway in the RED; the overall results of the MOE and cancer risk evaluations; and identification of any additional data that would prove useful in reducing the uncertainties of the MOE and cancer risk.

Table 9. Summary of the Occupational/Nonoccupational Exposure Scenarios Data

Exposure Scenario	Source of Data
Occupational Handler	
(1a) Mixing/Loading/Applying Liquids at a Pressure Treatment Facility (treatment operator)	Exposure Study Data from Creosote Council II and PHED, 1997used as a surrogate
(1b) Mixing/Loading/Applying at a Pressure Treatment Facility (treatment assistant)	Exposure Study Data from Creosote Council II
Occupational Postapplication	
(1) cylinder area loader operator	Exposure Study Data from Creosote Council II
(2) cylinder area loader helper	Exposure Study Data from Creosote Council II
(3) checker	Exposure Study Data from Creosote Council II
(4) drip pad labor	Exposure Study Data from Creosote Council II
(5) load-out area loader operator	Exposure Study Data from Creosote Council II
(6) load-out area loader helper	Exposure Study Data from Creosote Council II
(7) load out area forklift operator	Exposure Study Data from Creosote Council II
(8) oil unloader	Exposure Study Data from Creosote Council II
(9) pressure treatment test borer	Exposure Study Data from Creosote Council II
(10) water treatment systems operator	Exposure Study Data from Creosote Council II
(11) railroad worker	No data
(12) pole installer	No data
Non-Occupational (e.g., Residential)	
(1) homeowner incidental ingestion and dermal contact with soil contaminated with creosote (e.g., soil contaminated by creosote treated telephone poles) (child)	No data
(2) outdoor homeowner dermal contact with industry pressure treated wood products (e.g., utility poles, posts, shingles, fencing, lumber, piers, etc.) (adult)	No data
(3) outdoor homeowner hand-to-mouth and dermal contact with industry pressure treated wood products (e.g., utility poles, posts, shingles, fencing, lumber, piers, etc.) (child)	No data

In summary, the handler and post application MOEs and cancer risk are presented below:

Occupational Handler

- Scenario 1a exceeds long-term dermal MOE as well as the inhalation MOE and cancer risk criteria.
- Scenario 1b exceeds only inhalation MOEs and cancer risk criteria.

Occupational Postapplication

- Scenario 1 exceeds the long-term dermal and inhalation MOEs along with the cancer risk criteria.
- Scenario 2 exceeds intermediate- and long-term dermal and inhalation MOEs along with the cancer risk criteria.
- Scenario 3 exceeds intermediate- and long-term dermal and inhalation MOEs along with the cancer risk criteria.
- Scenario 4 exceeds intermediate- and long-term dermal and inhalation MOEs along with the cancer risk criteria.
- Scenario 5 exceeds the long-term dermal and inhalation MOEs along with the cancer risk criteria..
- Scenario 6 exceeds the inhalation MOE and cancer risk criteria.
- Scenario 7 exceeds the long-term dermal and inhalation MOEs along with the cancer risk criteria.
- Scenario 8 exceeds all MOE and cancer risk criteria.
- Scenario 9 exceeds the long-term dermal and inhalation MOEs along with the cancer risk criteria.
- Scenario 10 exceeds the inhalation MOE and cancer risk criteria.
- Scenario 11 There are no data for railroad worker exposure.
- Scenario 12 There are no data for utility pole installers.

Non-occupational Post-application

No data were submitted to characterize the residential scenarios. Site specific exposure data would be helpful to rectify the lack of data for the following occupational post-application scenarios.

- incidental ingestion and dermal contact with soil contaminated with creosote (e.g., soil contaminated by creosote treated telephone poles) (child)

- hand-to-mouth and dermal contact with pressure treated wood products (e.g., utility poles, posts, shingles, fencing, lumber, piers, etc.) (adult)

4.6 References

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